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Relationship of the type of tobacco and inhalation pattern to pulmonary and total mortality

P. Lange, J. Nyboe, M. Appleyard, G. Jensen, P. Schnohr

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ABSTRACT: Data from The Copenhagen City Heart Study, a prospective population study, were analysed to investigate the influence of the type of tobacco and inhalation on pulmonary and total mortality.

The study sample comprised 6,511 men and 7,703 women, selected randomly after age-stratification from the general population. There were 2,986 plain cigarette smokers, 3,222 filter cigarette smokers, 1,578 smokers of cheroots/cigars, 433 male pipe smokers and 773 subjects smoking more than one type of tobacco.

From 1976 until the end of 1989, 2,765 subjects died. Lung cancer was considered as main death cause in 268. Chronic obstructive pulmonary disease (COPD) was considered as the main cause in 94 cases and main or contributory cause of death in 195 cases (COPD related mortality). Current smokers had a higher risk of total mortality compared to lifetime nonsmokers: the relative risks (RR) ranged between 1.2 for male pipe smokers and 2.4 for female plain cigarette smokers. With regard to lung cancer mortality, the RR ranged between 4.1 for male pipe smokers and 7.9 for female plain cigarette smokers. Even higher RR values were estimated for COPD related mortality.

In both sexes, the RR for the investigated end-points were lower in cheroot/cigar smokers and in pipe smokers than in cigarette smokers, but these differences were markedly diminished after an adjustment for the inhalation habit.

The present study substantiates the view that tobacco smoking increases pulmonary and total mortality. The small differences between the various types of tobacco are probably caused by different inhalation patterns.

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In western countries, tobacco smoking is the principal cause of death from lung cancer and from chronic obstructive pulmonary disease (COPD) [1, 2]. In many large surveys, the relative risks of mortality from lung cancer and COPD in cigarette smokers compared to lifetime nonsmokers have been estimated [3]. However, relatively few studies have analysed possible differences between the effects of different kinds of smoking tobacco [4, 5], the main reason being that even large epidemiological studies often comprise relatively few persons smoking other kinds of tobacco than cigarettes.

Recently, a Swedish study of more than 25,000 male smokers estimated that mortality in pipe and cigar smokers may be as high as in cigarette smokers [6]. This observation is in contrast with earlier US and UK studies. The Swedish investigators suggested that the very high mortality risk in Swedish pipe smokers may be caused by the high prevalence of inhalation [6]. The purpose of the present investigation was to analyse mortality from primarily lung cancer and COPD. As the inhalation pattern may be of importance to the effect of smoking on mortality, this was also included in the analyses. Because there are several problems with the determination of the exact cause of death, especially in the case of deaths from non-neoplastic lung diseases [7], we included analyses of total mortality and the so called "COPD-related mortality", *i.e.* all deaths where COPD was listed on the death certificate as either the main or contributory cause of death.

Methods

Population and registration of causes of death

In 1976, a prospective epidemiological study, The Copenhagen City Heart Study, was initiated. The participants were selected among 90,000 persons living in

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19,698 subjects, aged 20 yrs or more, was selected at random. The sample fraction was highest (50%) for persons aged 40–69 yrs. A total of 14,223 subjects (74% of those invited) attended. Details on the selection procedure and a description of the nonresponders have been given previously, together with the questionnaire and the complete examination programme [8, 9].

The population sample described in the present paper comprised 6,511 men and 7,703 women. The nine female pipe smokers were excluded as the number of end-points in this group was too small for separate analysis. The subjects were followed until the end of 1989. Notification of deaths and causes of death was obtained from the Central Death Register from the National Board of Health. In Denmark, a physician, preferably the one who knew the patient's course best, must complete a death certificate at every death. The death certificate contains the assumed causes of death: always the underlying cause and in many cases also a secondary and a tertiary cause. The underlying cause is defined as a disease or injury which initiated the train of events leading directly to death, whereas the secondary or tertiary causes describe a disease (often a disease which may follow the underlying cause) which in some way could have contributed to death. The coding of deaths is performed centrally in the Death Register, based on the information stated on the death certificates. Death from lung cancer (code no. 162) was registered according to the 8th revision of the International Classification of Diseases (ICD) classification, which is still used in Denmark [10]. As the number of deaths from COPD was small in different smoking groups after stratification for sex, we mainly analysed the COPD-related mortality, which comprised the cases where chronic bronchitis or emphysema (codes no. 490–492) were considered as the underlying or as a contributing cause of death.

Smoking habits

The participants reported whether they were current smokers, ex-smokers or lifetime nonsmokers. Both current and ex-smokers reported the duration of smoking, whereas only current smokers reported their daily tobacco consumption, the type of tobacco smoked, and whether they inhaled or not. The subjects were not asked if they had smoked different types of tobacco in the past. No information on the depth of inhalation was obtained. The following groups of smokers were generated for the analyses:

NS: lifetime nonsmokers - subjects who at the enrolment reported that they had never smoked tobacco;
EX: ex-smokers - former smokers who ceased smoking more than one year prior to the examination;
CIGP: subjects smoking only plain (non-filter) cigarettes at enrolment;
CIGF: subjects smoking only filter cigarettes at enrolment;
CER: subjects smoking only cheroots, cigars or cigarillos at enrolment;
PIPE: men smoking only a pipe at enrolment;
MX: subjects who smoked more than one type of tobacco at enrolment.

To estimate the magnitude of current tobacco consumption, the following equivalents for the tobacco content were used: one plain or filter cigarette = 1 g; one cigar = 5 g; one cheroot or cigarillo = 3 g; one g of pipe tobacco = 1 g. For subjects who smoked more than one type of tobacco, the total amount of tobacco smoked per day was chosen as a measure of current tobacco consumption. For current smokers, an estimate of lifetime tobacco consumption was calculated as pack-years (current tobacco consumption ($g \cdot day^{-1}$) multiplied by the duration of smoking (yrs) and divided by 20).

Statistical analysis

The subject of the analysis was the intensity or instantaneous risk of death. We used the regression model of Cox [11]. Time was reckoned from the initial examination until the investigated endpoint (event), death from other causes (censoring) or until the end of the observation period. The regression coefficients were estimated using the maximum likelihood method as suggested by Cox. Smoking groups and inhalation were included as binary variables, as they only had two outcomes, whereas age was included directly as a quantitative variable.

The question of first order interactions between the covariates was investigated by comparison of regression models with and without interaction terms using the maximum likelihood ratio test. No significant interactions were found.

Control of the assumption of proportional hazards through-out the follow-up period was performed by plotting the logarithm to the cumulated hazard in different subgroups (strata) against time of observation. The distance between the curves in different smoking groups appeared to be the same throughout the period for all causes of mortality. For COPD-related mortality and lung cancer the curves for current smokers were parallel. However, the mortality in ex-smokers seemed relatively lower towards the end of the observation period, but because of the small number of deaths in this group a detailed analysis was not possible.

Separate regression models were employed for the three investigated end-points: death from all causes, death from lung cancer and COPD-related death. In all analyses, age was included as covariate and in the analyses comprising current smokers only, pack-years were also included in the model. Separate regressions were performed for men and women. In men, all the previously defined smoking groups could be analysed, whereas, in women, a sufficient number of end-points was only observed in the following groups: NS, EX, CIGP, CIGF and CER.

In the tables, the estimated relative risks (RR) and the 95% confidence intervals (95% CI) are given. If the 95% CI includes 1.0, the RR in this smoking group is not significantly different from the baseline group at the 5% level.

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On the basis of the RR in various groups of smokers relative to lifetime nonsmokers, we have calculated the population attributable risk of smoking, that is the proportion of the risk that may be attributed to smoking in subgroups of the population [12].

Table 1. – General characteristics of the subjects according to smoking group

	Women						Men						
Smoking gp	NS	EX	CIGP	CIGF	CER	MX	NS	EX	CIGP	CIG	CER	PIPE	MX
n	2159	1089	1344	2269	770	72	721	1253	1642	953	808	433	701
Age yrs	54	54	51	49	60	50	49	56	52	48	59	54	53
	(13)	(12)	(10)	(11)	(10)	(13)	(15)	(12)	(11)	(12)	(11)	(12)	(12)
Pack yrs	0	0*	19	15	25	23	0	0*	29	24	40	28	39
	(0)	(0*)	(11)	(11)	(16)	(17)	(0)	(0*)	(18)	(16)	(26)	(20)	(22)
Smoking	0	16	28	24	29	26	0	25	34	29	39	35	35
duration yrs	(0)	(12)	(10)	(12)	(13)	(12)	(0)	(14)	(12)	(12)	(14)	(14)	(13)
Inhalation %	0	0*	85	77	25	56	0	0*	93	91	42	56	75

Inhalation is given as a frequency whereas the other variables are given as arithmetical means. For definition of smoking groups see text. Data for age, pack-years and smoking duration are given as mean with SD in parenthesis. *: no information available regarding earlier inhalation pattern.

Table 2. – Mortality according to the smoking habit and sex

	Women						Men						
Smoking gp	NS	EX	CIGP	CIGF	CER	MX	NS	EX	CIGP	CIGF	CER	PIPE	MX
n	2159	1089	1344	2269	770	72	721	1253	1642	953	808	433	701
Mortality													
Total	262	133	224	241	185	10	115	318	476	206	326	115	154
(%)	(12)	(12)	(17)	(11)	(24)	(14)	(16)	(25)	(29)	(22)	(40)	(27)	(22)
Lung cancer	7	8	20	19	14	0	5	21	65	25	47	16	21
(%)	(0.3)	(0.7)	(1.5)	(0.8)	(1.8)	(0)	(0.7)	(1.7)	(4.0)	(2.6)	(5.8)	(3.7)	(3.0)
COPD	2	6	6	13	4	0	2	14	24	12	4	2	5
%	(0.1)	(0.6)	(0.4)	(0.6)	(0.5)	(0)	(0.3)	(1.1)	(1.5)	(1.3)	(0.5)	(0.5)	(0.7)
COPD-related	3	11	12	22	10	1	3	25	43	23	22	7	13
%	(0.1)	(1.0)	(0.9)	(1.0)	(1.3)	(1.4)	(0.4)	(2.0)	(2.6)	(2.4)	(2.7)	(1.6)	(1.9)

For definition of smoking groups see text. %: percentage of all subjects in the smoking group. COPD: chronic obstructive pulmonary disease.

Results

The distribution of the subjects according to sex and smoking group at the time of enrolment in the study is given in table 1. On average, men and women smoking cheroots/cigars were older and had a higher cumulative tobacco consumption than other smokers. Plain cigarette smokers reported inhalation more frequently than did smokers in the other groups.

The distribution of deaths by sex and smoking group is given in table 2. Generally, the frequency of all investigated end-points was higher in smokers than in lifetime nonsmokers, regardless of the type of tobacco smoked. Similar trends were observed in both sexes.

The results of the Cox regression analysis focusing on the risk of total and pulmonary mortality in smokers smoking different types of tobacco, compared to the risk in lifetime nonsmokers, are given in table 3. The risk of death increased significantly with age. The relative risks (RR) of total and pulmonary mortality in all smoking groups were >1. Generally, the RR were higher in women than in men, especially with regard to COPD related mortality.

Table 3. – Relative risks (RR) of mortality by smoking status with NS as the baseline group

Smoking group	Total mortality RR (95% CI)	Lung cancer mortality RR (95% CI)	COPD-related mortality RR (95% CI)
Women			
NS	1.0	1.0	1.0
EX	1.2 (1.0–1.4)	3.4 (1.1–11)	11 (2.5–53)
CIGP	2.4 (2.0–2.9)	7.9 (2.9–21)	15 (3.1–65)
CIGF	1.7 (1.4–2.1)	4.8 (1.7–13)	16 (3.6–70)
CER	1.8 (1.4–2.2)	4.9 (3.0–12)	10 (2.3–48)
Men			
NS	1.0	1.0	1.0
EX	1.1 (0.9–1.4)	2.1 (0.7–6.0)	3.0 (0.9–10)
CIGP	1.9 (1.6–2.4)	7.3 (2.6–20)	6.4 (2.0–20)
CIGF	1.8 (1.4–2.3)	6.0 (2.2–19)	7.9 (2.3–27)
CER	1.6 (1.3–2.0)	6.0 (2.2–17)	3.7 (1.1–12)
PIPE	1.2 (0.9–1.6)	4.1 (1.4–13)	2.4 (0.6–9.6)
MX	1.2 (0.9–1.6)	4.5 (1.6–13)	4.3 (1.2–15)

Derived from regression models of Cox including age as covariate. NS: lifetime nonsmokers; For definition of smoking groups see text. 95% CI: 95% confidence interval; COPD: chronic obstructive pulmonary disease.

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The differences in mortality between smokers of plain cigarettes and those smoking other types of tobacco, after adjustment for age and pack-years, are shown in table 4. With regard to total mortality the RR in plain cigarette smokers was higher than in the other groups, especially in women. With regard to pulmonary mortality, none of the differences between plain and filter cigarette smokers were statistically significant.

In women the RR in cheroot smokers were 30–40% reduced compared to smokers of plain cigarettes (table 4).

Table 4. – Relative risks (RR) of mortality in current smokers by the type of tobacco smoked

Smoking group	Total mortality RR (95% CI)	Lung cancer mortality RR (95% CI)	COPD-related mortality RR (95% CI)
Women			
CIGP	1.0	1.0	1.0
CIGF	0.7 (0.6–0.9)	0.7 (0.4–1.4)	1.3 (0.6–1.6)
CER	0.7 (0.6–0.9)	0.6 (0.4–1.3)	0.7 (0.3–0.7)
Men			
CIGP	1.0	1.0	1.0
CIGF	0.9 (0.8–1.1)	0.9 (0.6–1.4)	1.2 (0.7–2.0)
CER	0.8 (0.7–0.9)	0.7 (0.5–1.1)	0.5 (0.3–0.8)
PIPE	0.6 (0.5–0.8)	0.6 (0.3–1.0)	0.4 (0.2–0.8)
MX	0.6 (0.5–0.7)	0.3 (0.1–1.4)	0.2 (0.1–0.6)

Derived from regression models of Cox including age and pack-years as covariates. For definition of smoking group see text. For abbreviations see legend to table 3.

In men, the RR values in pipe and in mixed smokers (MX) were 40–80% reduced compared with cigarette smokers, whereas the RR in cheroot/cigar smokers was inbetween the risk of cigarette smokers and the risk of pipe smokers.

To investigate the importance of reported inhalation pattern on mortality, the following grouping of smokers was employed: cigarette smokers (both plain and filter) who reported inhalation, cigarette smokers who reported no inhalation, smokers of other types of tobacco than cigarettes (cheroot/cigar and pipe) who reported inhalation, and finally smokers of other types of tobacco than cigarettes who reported no inhalation. The RR values of total and pulmonary mortality for these four groups are shown in table 5. Both cigarette and non-cigarette smokers who reported inhalation had a higher risk of death from the investigated end-points than smokers who said they did not inhale. The RR for the investigated end-points for women smoking cheroots and inhaling was significantly higher than for female cigarette smokers who inhaled.

Based on the RR given in table 3 and on the distribution of the different smoking groups we have calculated the attributable risks shown in table 6. The proportion of death that could be attributed to smoking was 0.36 in both sexes. With regard to lung cancer and COPD-related deaths the proportion was more than twice as high. Generally plain cigarette smoking was the highest contributing smoking modality to the deaths from the investigated diseases. However, in women the contribution of filter cigarette smoking to COPD-related mortality was the highest.

Table 5. – Relative risks (RR) of mortality in current smokers by the type of tobacco smoked and inhalation

Smoking group	Total mortality RR (95% CI)	Lung cancer mortality RR (95% CI)	COPD-related mortality RR (95% CI)
Women			
CIGP+CIGF + inhalation	1.0	1.0	1.0
CIGP+CIGF - inhalation	0.7 (0.6–0.9)	0.3 (0.1–0.8)	0.4 (0.2–1.1)
CER + inhalation	1.6 (1.2–2.2)	1.5 (0.5–3.7)	2.1 (0.8–5.3)
CER - inhalation	0.6 (0.5–0.8)	0.4 (0.2–0.9)	0.2 (0.1–0.6)
Men			
CIGP+CIGF + inhalation	1.0	1.0	1.0
CIGP+CIGF - inhalation	0.9 (0.7–1.2)	0.2 (0.1–0.8)	0.5 (0.2–1.4)
CER+PIPE + inhalation	1.0 (0.8–1.2)	1.1 (0.7–1.6)	0.9 (0.5–1.6)
CER+PIPE - inhalation	0.7 (0.6–0.8)	0.4 (0.3–0.6)	0.2 (0.1–0.3)

Derived from regression models of Cox including age and pack-years as covariates. For definition of smoking groups see text. For abbreviations see legend to table 3.

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Table 6. – Estimated proportion of the investigated end-points attributable to smoking and contribution from individual smoking groups

Smoking group	Total mortality		Lung Cancer mortality		COPD-related mortality	
	Women	Men	Women	Men	Women	Men
Attributable proportion to smoking*	0.36	0.36	0.75	0.79	0.90	0.78
Contribution from Subgroups						
EX	0.02	0.02	0.08	0.04	0.15	0.09
CIGP	0.15	0.15	0.29	0.33	0.23	0.30
CIGF	0.14	0.08	0.28	0.17	0.43	0.22
CER	0.05	0.09	0.10	0.13	0.09	0.07
PIPE		0.01		0.04		0.02
MX		0.01		0.08		0.08

For definitions of smoking groups see text.*: including ex-smokers. COPD: chronic obstructive pulmonary disease.

Discussion

We found that smokers, regardless of the type of tobacco smoked, had a higher risk of total and pulmonary mortality than lifetime nonsmokers. This pertained to both men and women. After adjustment for cumulated tobacco consumption, the differences in the mortality risk between plain and filter cigarette smokers were small and statistically significant only in the case of total mortality in women. Compared with cigarette smokers, smokers of cheroots/ cigars and especially pipe smokers had a lower risk of mortality, although the risk was still markedly higher than in lifetime nonsmokers. Both cigarette and non-cigarette smokers who said they inhaled had a higher risk of mortality than smokers who did not inhale.

As for many previous studies, our study suffers from several limitations. The information on lifetime tobacco consumption is incomplete, as we did not ask the participants what kind of tobacco they smoked before enrolment into the study. This information may be especially important in pipe and cheroot/cigar smokers who have previously been smoking cigarettes [5]. We were also unable to include changes in smoking habits during the follow-up in the analyses. Another drawback of the present study is the small number of deaths from COPD in many of the smoking groups. To enlarge the number of end-points, we also analysed the COPD-related deaths. An earlier study has indicated that this approach may be correct [13].

In spite of these apparent limitations, the present study also has advantages: an acceptable attendance of 73.6% of the invited subjects; the very high follow-up rate as only 63 subjects left Denmark and only one was lost during the observation period; the relatively high frequency of smoking other types of tobacco than cigarettes; and, finally, in contrast to the study of CARSTENSEN *et al.* [6], information on the inhalation habits of pipe and cheroot/cigar smokers.

Plain and filter cigarettes compared

Compared with lifetime nonsmokers, cigarette smokers had the highest RR of all the investigated end-points. The RR of total mortality, lung cancer and COPD mortality are similar to earlier results [1–3]. After statistical adjustment for the cumulative tobacco consumption, the RR of pulmonary and total mortality in plain cigarette smokers and filter cigarette smokers were very similar in men. Female filter cigarette smokers had a 30% lower risk of all-causes of mortality and lung cancer mortality, whereas they had a 30% higher risk of COPD-related mortality than smokers of plain cigarettes. Previous studies, have shown that subjects smoking cigarettes with lower tar yields may have a risk of lung cancer which is 5–50% lower than subjects smoking cigarettes with higher tar yields, depending on the study [3, 14, 15]. It is possible that the relatively small difference in lung cancer mortality observed in the present study might be due to the relatively small difference in the tar yields of the cigarettes compared: at the time of our study, the average tar content in Danish cigarettes was 35 mg per cigarette in plain cigarettes and 23 mg per cigarette in filter cigarettes.

The risk of death from COPD is closely related to the level of lung function [13, 16]. As most previous studies have been unable to show a significant benefit from smoking lower-tar cigarettes, compared to high- and medium-tar cigarettes, on lung function and lung function decline [17–19], our results on COPD-related mortality support the theory that tar content of the cigarettes may be relatively unimportant for COPD [17]. In contrast to the observations of The American Cancer Society study [4], the trend observed in the present study suggests that smokers of filter cigarettes may even have a slightly higher risk of COPD than smokers of plain cigarettes.

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Cigar/cheroot and pipe smoking

Compared with lifetime nonsmokers, the estimated RR for mortality was 1.6–1.7 for cheroot/cigar smokers and 1.2 for pipe and mixed smokers. These risks are higher than the values reported by HAMMOND [4] in 1966 in a US population. This may be partly explained by the fact that the US analysis was performed on smokers who had previously only been smoking cigars or pipes. As in earlier European studies, the mortality from lung cancer in cheroot/cigar and pipe smokers in the present study was much higher than in lifetime nonsmokers and only slightly lower than in cigarette smokers [5, 6, 15, 20]. This is in accordance with the experimental results, which show that condensates from pipes and cigars are at least as carcinogenic as condensates from cigarettes [3].

With regard to COPD, the differences between cigarette and non-cigarette smokers were more pronounced than in the case of the lung cancer. The risk of COPD-related mortality in cheroot/cigar and pipe smokers was reduced by approximately 30–50% as compared with cigarette smokers, but it was still much higher than in lifetime nonsmokers. In the study of British doctors, the risk of COPD mortality in pipe and cigar smokers was approximately 10 times higher than in lifetime nonsmokers, although it was only one third of the risk of cigarette smokers [5].

Inhalation

Subjects who reported inhalation had a higher mortality from all of the investigated end-points than those who said they did not inhale. This was especially the case for pulmonary mortality. After adjustment for the inhalation pattern, the differences in mortality between smokers of different types of tobacco were markedly diminished. Thus, not surprisingly, our results indicate that the inhalation of tobacco smoke should be avoided.

With regard to the importance of inhalation to total mortality, the large American Cancer Society Study agrees with our findings [4]. With regard to pulmonary mortality, the importance of inhalation has in the past been subject to some controversy. DOLL and PETO [5] reported a positive association between mortality from COPD and inhalation of tobacco smoke, but a surprising negative correlation between inhalation and lung cancer mortality. The latter finding has been reproduced in another UK survey [21]; however, GARFINKEL and STELLMAN [22] using data from the American Cancer Society Study II reported a higher risk of lung cancer with an increasing degree of inhalation. The discrepancies between various studies suggest that the value of reported inhalation as an index of actual inhalation may be low, especially in cigarette smokers who comprised the vast majority of subjects in the UK surveys. This has also been shown experimentally with measurements of biological inhalation markers [23]. On the other hand, previous studies have shown that the value of self-reported inhalation in pipe and cigar smokers may in fact be quite high and that this information may be even more useful than the knowledge of whether the current non-cigarette smokers had been smoking cigarettes in the past [24, 25]. Our results show that information on inhalation is valuable even if it is not validated by biological markers.

Male-female differences

An analysis focusing on the differences in RR values between sexes (not shown), revealed that the differences were not statistically significant. In Denmark, as in some other European countries, smoking is steadily declining in men, but much less so in women. The latest Danish data show that in young subjects aged 15–20 yrs, smoking is even more prevalent in women than in men [26]. If this trend continues in the future, it is likely that diseases such as lung cancer and COPD will become equally common in both sexes.

Attributable risks

The proportion of the investigated deaths attributable to smoking in the present study is very close to the results of the American Cancer Society 50 State Study [3]. The slightly higher proportion of lung cancer cases in men than in women that could be attributed to smoking is in line with observations that lung cancers in lifetime nonsmokers are more often seen in women than in men [27]. On the other hand, we observed a higher proportion of COPD-related deaths in women than in men that could be attributed to smoking. This could reflect the possible role of occupational exposures to dusts and fumes in men, although differences in the diagnostic criteria of COPD between the sexes could also be responsible for our results.

A calculation of the attributable risks in the subgroups of smokers showed that most of the smoking-related deaths in men could be attributed to plain cigarette smoking. In women, however, filter cigarettes "were responsible" for more COPD-related deaths than plain cigarettes. This reflects the fact that women, more often than men, smoked filter cigarettes and that the RR for COPD-related mortality were higher in filter cigarette smokers than in plain cigarette smokers. Although we found a surprisingly high RR for cheroot/cigar and pipe smokers, the contribution of these types of tobacco to mortality was small, as relatively few subjects smoked these tobacco types. It is likely that this proportion will be even smaller in the future, as smoking cessation rates in this subgroup of smokers are higher than in cigarette smokers [26].

In conclusion, this study shows that although cigarette smokers are at highest risk of death from lung

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cancer and COPD, the differences between cigarette smokers and smokers of other types of tobacco are probably due to different inhalation patterns, and are relatively small compared to the differences between the latter group and nonsmokers.

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